Passive smoking, salivary cotinine concentrations, and middle ear effusion in 7 year old children

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Abstract

Objective—To assess the contribution of passive exposure to tobacco smoke to the development of middle ear underpressure and effusion.

Design—Cross sectional observational study.

Setting—One third of the primary schools in Edinburgh.

Subjects—892 Children aged $6^{1/2}$ to $7^{1/2}$ were examined, and satisfactory tympanograms were obtained in 872. Results of assay of salivary cotinine concentrations were available for 770 children, and satisfactory tympanograms were available for 736 of these.

End point—Correlation of the prevalence of middle ear underpressure and effusion with concentrations of the marker of nicotine, cotinine, in the saliva of the children.

Measurements and main results—Middle ear pressure and compliance were measured in both ears by impedance tympanometry. Salivary cotinine concentrations were assayed by gas-liquid chromatography. Cotinine concentrations increased with the number of smokers in the household. Girls had higher concentrations than boys, and children living in rented housing had higher concentrations than those living in housing owned by their parents. There was a trend towards more abnormal tympanometric findings with increasing cotinine concentration, the odds ratio for a doubling of the cotinine concentration being 1·14 (95% confidence interval 1·03 to 1·27). After adjustment for the sex of the child and housing tenure the odds ratio for a doubling of the cotinine concentration was 1·13 (1·00 to 1·28).

Conclusions—The results of this study are consistent with those of case-control studies of children attending for an operation to relieve middle ear effusion. They indicate that the disease should be added to the list of recognised hazards associated with passive smoking. About one third of the cases of middle ear effusion in this study were statistically attributable to exposure to tobacco smoke.

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Introduction

Middle ear effusion (glue ear) is the commonest reason for admitting young children for an operation,[1] but little is known about its cause.[2] Case-control studies of children admitted for insertion of a grommet have shown an increased risk associated with the presence of smokers in the household,[3-5] particularly in children who had been exposed to high levels of tobacco smoke.[3] Criteria for referral and admission for middle ear effusion seem to be determined substantially by the "health culture" of the family[4] and by local clinical practice, which may in turn be affected by the availability of services.[3] Thus the interpretation of studies based on patients in hospital is complicated by selection bias, which may result in either a spurious relation with parental smoking or an underestimate of a true effect.

Five studies of children in the general population have reported on the association between middle ear effusion and passive exposure to smoke.[6-12] Only Iversen *et al* found a significant association, and their results suggested that the risk associated with passive smoking increased with age.[8] If this is so it might explain the negative results of the other studies, which were based on children aged less than 5 years old.[6-12]

Both middle ear effusion and exposure to tobacco smoke can be measured objectively. Since its introduction some 20 years ago[13] impedance tympanometry has been widely used as a diagnostic and screening tool in young children, and its relation to fluid in the middle ear has been validated in patients attending for myringotomy.[14,15] Cotinine, an important metabolite of nicotine, is the most suitable marker to measure passive exposure to tobacco smoke. It is specific, has a half life of about 20 hours, and can be assayed in concentrations as low as 0·57 nmol/l (0·1 ng/ml) by gas-liquid chromatography.[16] Salivary concentrations of cotinine are roughly in proportion to those in blood and have been used to measure exposure to environmental tobacco smoke in adults[17,18] and adolescents.[19,20]

We investigated the relation between exposure to smoke and middle ear disease in a sample population of 7 year old schoolchildren who were participating in a survey of the effects of the home environment on respiratory health.[21]

Subjects and methods

A sample of one in three primary schools in Edinburgh was chosen at random, and the parents of all children in the third primary class (aged 6½–7½ years in September 1986) were contacted by postal questionnaire. This asked about respiratory symptoms and housing conditions relating to the child; more details are reported elsewhere.[21] The current or latest occupation of the head of the household was coded to a social class according to the registrar general's classification of occupations.[22] Written parental consent to clinical tests was requested, and ethical approval for the study was obtained from Lothian Health Board and Lothian Regional Council's education department.

Clinical tests were performed at the schools during January to June under the supervision of DPS. Middle ear pressure and compliance, the volume of the ear canal, and the relative gradient of the tympanometric curve were measured in both ears with a Microlab Earscan configured for impedance measurements (Micro Audiometrics, Florida, United States). This uses a probe tone of 226 Hz at 85 db and sweeps from 200 to −312 daPa at 100 daPa/s. The children were asked to swallow a sip of water immediately before the measurement was made to ensure that patent eustachian tubes would be ventilated. Table I shows the types of tympanograms, defined on the basis of the modification of Jerger's original classification[23] that was proposed

TABLE I—*Types of tympanograms according to Fiellau-Nikolajsen*[24]

Type	Middle ear pressure (daPa)	Gradient (%)	Interpretation
A	200 to −99·9	>10	Normal
C1	−100 to −199·9	>10	Mild underpressure
C2	−200 to −312	>10	Severe underpressure
B	No peak	<10	Middle ear effusion

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and validated by Fiellau-Nikolajsen.[1] To characterise each child the tympanogram obtained on examination of the more abnormal ear was used in the analysis.

The children were asked to collect saliva in their mouths and to spit into a clean plastic container. A sample of at least 1 ml was frozen within eight hours after collection for assay of cotinine concentration by gas-liquid chromatography.[16] Statistical analyses were performed by the statistical analysis system (SAS),[24 25] and logistic regression models were fitted by the generalised linear interactive modelling system (GLIM).[26] Tests for trend with one degree of freedom used the procedure proposed by Mantel[27] and implemented for stratified tabulations in the FREQ procedure in SAS.[25]

Results

The parents of 1095 children were sent a questionnaire, and 941 (86%) consented to their child being examined clinically. Twenty of these children left school before the survey was carried out, and a further 20 were included in pilot studies. Of the remaining 901 eligible children, 892 (99%) were examined, and satisfactory tympanograms were obtained for one or both ears in 872. In 23 children results were obtained for only one ear, but these were included in the analysis.

Table II shows the relation between findings on tympanometry and the sex of the child, social class, housing tenure, and number of smokers in the household. There was no significant difference in middle ear pressure with sex or social class (when this was known), but the prevalence of middle ear effusion (type B tympanogram) was higher in girls and among children of unknown social class. There was a significant trend towards abnormal tympanograms in the children whose parents were smokers, and the prevalence of effusion increased with the number of smokers in the household (χ^2 for trend in proportions = 4·15, df = 1, $p < 0·05$). There was a similar trend, which was of borderline significance ($p = 0·051$), associated with rented housing.

The results of the salivary cotinine assay were available for 770 children (405 from non-smoking households, 241 from households with one smoker, and 124 from households with two or more smokers). Table III shows the distributions of salivary cotinine concentrations in these three groups. A total of 109 (27%) children from households with no smokers had concentrations below the limit of the assay (0·57 nmol/l), whereas only one child from a household with one or more smokers had no detectable salivary cotinine. Six children, five of them from households with only one smoker, had concentrations >82 nmol/l, a suggested cut off point to distinguish between smoking and non-smoking adults and adolescents.[28 29] These values were 93·1, 97·1, 106·2, 119·3, 144·8, and 205·0 nmol/l.

Table IV shows the relation between cotinine concentrations, sex of the children, and housing tenure within groups with similar numbers of smokers in the home.

TABLE II—*Prevalence (%) of types of abnormalities on tympanometry of the more abnormal ear in 872 children aged 7 according to sex of child, socioeconomic state, housing tenure, and number of smokers in household. Numbers of children are given in parentheses*

	Middle ear pressure (daPa)				
	100 to −100 (type A)	−200 to −100 (type C1)	−300 to −200 (type C2)	No peak (type B)	χ^2 Trend*
Sex:					
Girls	63·1 (275)	17·0 (74)	12·4 (54)	7·6 (33)	
Boys	62·2 (271)	17·2 (75)	9·4 (41)	11·2 (49)	0·60
Social class of head of household:					
I	63·9 (62)	16·5 (16)	12·4 (12)	7·2 (7)	
II	63·9 (145)	14·5 (33)	14·5 (33)	7·1 (16)	
IIIN	62·4 (108)	21·4 (37)	5·2 (9)	11·0 (19)	0·00
IIIM	65·2 (118)	17·7 (32)	6·6 (12)	10·5 (19)	
IV/V	62·9 (66)	14·3 (15)	17·1 (18)	5·7 (6)	
Unknown†	52·8 (47)	18·0 (16)	12·4 (11)	16·9 (15)	
Tenure of housing:					
Owned	64·6 (396)	16·3 (100)	10·6 (65)	8·5 (52)	
Rented	57·8 (147)	18·5 (47)	11·8 (30)	11·8 (30)	3·80
No of smokers in household:					
0	63·9 (292)	17·3 (79)	10·7 (49)	8·1 (37)	
1	63·3 (169)	16·5 (44)	10·9 (29)	9·4 (25)	3·97‡
≥2	56·4 (79)	17·1 (24)	12·1 (17)	14·3 (20)	
Fifths of salivary cotinine (nmol/l):					
<0·57	64·8 (70)	15·7 (17)	12·0 (13)	7·4 (8)	
0·57–	72·6 (130)	13·4 (24)	8·9 (16)	5·0 (9)	
2·27–	65·4 (104)	20·8 (33)	5·7 (9)	8·2 (13)	7·01§
7·38–	61·0 (89)	17·8 (26)	6·9 (10)	14·4 (21)	
>19·9	58·3 (84)	16·7 (24)	12·5 (18)	12·5 (18)	

* df = 1.
† Head of household was a student, a member of the armed forces, or had never been employed; this group was excluded from the test for linear trend.
‡ $p < 0·05$. § $p < 0·01$.

TABLE III—*Distribution of salivary cotinine concentrations according to number of smokers in household*

No of smokers in household	Salivary cotinine (nmol/l)				
	Minimum	First quartile	Median	Third quartile	Maximum
0 (n = 405)	ND	ND	1·1	2·3	36·9
1 (n = 241)	ND	4·5	10·2	22·7	205·0
≥2 (n = 124)	2·3	12·5	25·0	37·5	97·1
Total (n = 770)	ND	0·6	4·0	16·5	205·0

ND = None detected (<0·57 nmol/l).

TABLE IV—*Geometric mean salivary cotinine concentrations* (nmol/l) in 770 children aged 7 according to sex of child, housing tenure, and number of smokers in household. Numbers of children are given in parentheses*

No of smokers in household	Tenure of housing			
	Owned		Rented	
	Boys	Girls	Boys	Girls
0	0·85 (176)	1·02 (161)	3·01 (35)	8·63 (33)
1	5·62 (67)	6·53 (74)	17·04 (53)	25·95 (47)
≥2	15·61 (28)	17·04 (33)	21·47 (30)	34·36 (33)

* Undetectable concentrations were recorded as 0·28 nmol/l.

In view of the skewed nature of the distributions for cotinine concentrations the table gives geometric mean values. For logarithmic transformation undetectable concentrations were treated as 0·28 nmol/l. Female sex and rented housing were independently and consistently associated with higher cotinine concentrations given the same number of smokers in the household. These effects were apparent even in non-smoking households, and the difference with sex was particularly pronounced among children from rented homes.

Satisfactory tympanograms were obtained for 736 of the 770 children for whom we had data on salivary cotinine concentrations. When cotinine concentrations were grouped in fifths of the distribution there was a highly significant trend towards more abnormal tympanograms in the children with higher concentrations (table II). In view of the associations between cotinine concentrations and sex of the child and housing tenure and the modest effect of these factors on the prevalence of middle ear effusion the relation between salivary cotinine concentrations and abnormal tympanograms was analysed further by multiple logistic regression. Presence or absence of effusion (type B tympanogram) was treated as a dichotomous outcome variable. To investigate the form of the dose-response relation in more detail the data on cotinine concentrations were fitted as a continuous explanatory variable. The logarithm of the cotinine concentration was found to give the best fit, its relation to the prevalence of type B tympanograms being close to linear on a logarithmic scale (χ^2 for inclusion of quadratic term = 0·0000, df = 1).

In single factor models the odds ratio for female sex was 1·53 (95% confidence interval 0·92 to 1·98), and

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for rented housing it was 1·43 (0·84 to 2·42). The effect of the logarithm of the cotinine concentration in a single factor model was significant ($\chi^2=6·60$, df=1, $p<0·02$), and the odds ratio for a doubling of salivary cotinine concentration was 1·14 (1·03 to 1·27). In a joint model including all three factors the effects of sex and logarithm of the cotinine concentration changed little, but there was an appreciable reduction in the odds ratio for children living in rented housing, suggesting that passive exposure to smoke accounted for much of the effect of rented housing in the single factor models. The adjusted odds ratios were 1·46 (0·87 to 2·44) for female sex, 1·03 (0·55 to 1·91) for rented housing, and 1·13 (1·00 to 1·28) for a doubling of salivary cotinine concentration. The effect of the logarithm of the cotinine concentration remained significant in the joint model ($\chi^2=4·14$, df=1, $p<0·05$). Further adjustment for parental social class, number of people living in a room, gas cooking, and the presence of damp walls in the home made no substantial difference to the coefficient for the logarithm of the cotinine concentration.

The linear relation between the logarithm of the cotinine concentration and the prevalence of middle ear effusion on a logit scale implied that the prevalence odds were proportional to a power of the cotinine concentration, the power exponent being the coefficient (logarithm of the odds ratio) for the logarithm (base e) of the cotinine concentration in the logistic model. The data suggested that the odds ratios for type B tympanograms after adjustment for sex and housing tenure relative to children with undetectable cotinine concentrations would be approximately 1·7 at 5·7 nmol/l (1 ng/ml) and 2·3 at 28·4 nmol/l (5 ng/ml). Thus even low levels of passive exposure to smoke may have substantial effects on the prevalence of middle ear effusion. The model predicted that in a population of the same distributions of age, sex, and tenure in which all children had undetectable cotinine concentrations the prevalence of type B tympanograms would be approximately 5·8%. As the observed prevalence was 9·4% at least one third of the cases of middle ear effusion in this age group may have been attributable to passive smoking.

Discussion

We believe that this is the first study to report biochemical data on passive exposure to smoke in primary school children. The age group chosen was young enough to exclude regular active smoking, but some of the higher concentrations of salivary cotinine observed were greater than could reasonably have been attributed to passive exposure. These high concentrations may indicate experimentation with cigarettes, even at this early age. None of the six children with cotinine concentrations above 82 nmol/l, however, had middle ear effusion (five had normal (type A) tympanograms), so their inclusion in the analysis will have tended to diminish any effects attributed to passive exposure to smoke rather than to generate a spurious effect.

As expected, cotinine concentrations were related to the number of smokers in the household, but equally striking was the variation with sex of the children and housing tenure within groups with the same numbers of smokers. Even among the children from non-smoking households cotinine concentrations were higher in those living in rented accommodation. This suggests that considerable exposure to smoke occurs outside the home, which is strongly related to social factors. When tenure and number of smokers were controlled for girls had higher salivary cotinine concentrations than boys. This may reflect differences in cotinine metabolism or in activity patterns, boys being perhaps more likely to play outdoors or away from adults who smoke. No difference with sex has been found in older children.[16]

The prevalence of abnormalities on tympanometry in this population of 7 year old children is consistent with previous reports.[2,17] Tympanometry was performed only once, and many of the abnormalities detected, including middle ear effusion, tend to resolve spontaneously.[2] This population survey probably included only a few cases of persistent disease in which an operation would be indicated. The findings are therefore complementary to, rather than directly comparable with, case-control studies of children admitted to hospital.[1,2] They do, however, relate to an age group close to the peak age for admission for an operation for middle ear effusion.[1]

Our results show a significant relation between salivary cotinine concentrations and disease of the middle ear, whether a range of abnormal tympanograms, or tympanograms with and without a definable peak (taken to indicate effusion) are considered. These associations were probably not due to bias because the measurements were objective and the laboratory analysts were blind to the tympanometric findings. Adjustment of the crude estimates of the effect of sex and housing tenure on cotinine concentrations indicated some confounding by these factors, but confounding by socioenvironmental factors probably did not persist in the final model. The coefficient for housing tenure in the joint model was small, and further adjustment for social class and a range of more specific housing characteristics made little difference to the results.

Salivary cotinine concentrations relate only to passive exposure to smoke in the previous two or three days, but Jarvis *et al* reported that over one year concentrations in non-smoking adolescent girls was reasonably stable.[18] Nevertheless, variation in exposure from week to week limits the reliability of a single measurement, and the true association between passive smoking and middle ear effusion is therefore underestimated in our data.[11]

The relation between passive exposure to smoke and middle ear effusion in the present study is more likely to be causal than due to chance, bias, or confounding factors. The common mechanism in the development of serous otitis media is considered to be loss of patency of the eustachian tube, to which anatomical factors, impaired mucociliary function, and upper respiratory infection or allergy may contribute.[2] Passive smoking may increase the risk of blockage of the eustachian tube in three ways: by directly impairing mucociliary function; by causing congestion of the soft tissues of the nasopharynx; or by predisposing people to upper respiratory infection. Because this was a study of the prevalence of middle ear effusion we cannot draw conclusions about whether exposure to smoke influences the incidence or the persistence of the disease.

Concern has been expressed recently that the documented risks of passive smoking have not included middle ear effusion.[19] In view of the important burden on the health service imposed by this disease[1] and suspicions of its long term effects on linguistic and cognitive development[20] middle ear effusion in children should be regarded as one of the more important hazards attributable to environmental tobacco smoke.

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Relation between mortality and treated blood pressure in elderly patients with hypertension: report of the European Working Party on High Blood Pressure in the Elderly

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Abstract

Objective—To investigate the relation between mortality and treated systolic and diastolic blood pressures.

Design—Randomised double blind placebo controlled trial. Mortality in the two treatment groups was examined in thirds of treated systolic and diastolic blood pressures.

Patients—339 And 352 patients allocated to placebo and active treatment, respectively. The groups were similar at randomisation in sex ratio (70% women), mean age (71·5 years), blood pressure (182/101 mm Hg), and proportion of patients with cardiovascular complications (35%).

Measurements and main results—In the placebo group total mortality rose with increasing systolic pressure whereas it had a U shaped relation with diastolic pressure, the total lowest mortality being in patients in the middle third of the distribution of diastolic pressure. In the group given active treatment total mortality showed a U shaped relation with systolic pressure and an inverse association with treated diastolic pressure. In both groups cardiovascular and non-cardiovascular mortality followed the same trends as total mortality. The increased mortality in the lowest thirds of pressure was not associated with an increased proportion of patients with cardiovascular complications at randomisation or with a fall in diastolic pressure exceeding the median fall in pressure in each group. In contrast, patients in the lowest thirds of pressure showed greater decreases in body weight and haemoglobin concentration than those in the middle and upper thirds of pressure.

Conclusions—In patients taking active treatment total mortality was increased in the lowest thirds of treated systolic and diastolic blood pressures. This increased mortality is not necessarily explained by an exaggerated reduction in pressure induced by drugs as for diastolic pressure a U shaped relation also existed during treatment with placebo. In addition, patients in the lowest thirds of systolic and diastolic pressures were characterised by decreases in body weight and haemoglobin concentration, and the patients in the lowest thirds of diastolic pressure taking active treatment also by an increased non-cardiovascular mortality, suggesting some deterioration of general health.

Introduction

Several large studies of hypertension have recently been reviewed.[1,2] The observation in these studies of a J shaped relation between the risk of myocardial infarction and treated blood pressure[3-9] has led to the suggestion that a reduction of pressure induced by drugs might cause as well as prevent myocardial ischaemia.[1,10,11] None of the studies was placebo controlled, and other large hypertension-mortality intervention trials have either not confirmed[12-14] or not reported[15,16] this J shaped relation. In the international prospective primary prevention study in hypertension all patients received active drugs but patients with overt ischaemic heart disease were excluded[16]; there was no evidence for a J curve. In contrast, Coope and Warrender found that total mortality and deaths from myocardial infarction showed a J shaped relation with the diastolic pressure attained in elderly patients with

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Collaborating centres are listed at the end of this paper. Manuscript prepared by J Staessen and A E Fletcher

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undecided.[1] I found similar responses amongst a random sample of 1000 general practitioners in New Zealand: 22% reported that informed consent for testing was not at all important and 4% were undecided. I, too, found that years since graduation was a significant variable as was the number of patients requesting an HIV test. Younger doctors and those with the most requests for tests believed more strongly in informed consent.

In addition I examined attitudes to anonymous testing and to confidentiality. Anonymous testing, although recommended and widely practised in New Zealand, was disagreed with by 28% of the sample, including 7% who disagreed strongly. In fact, only 41% agreed with it, the remainder being neutral. Attitudes to confidentiality were shown by the doctors' responses to questions on sharing information about patients with AIDS. Sixteen per cent of the doctors would give such information to reception staff, and the same proportion would give it to colleagues outside the practice. Women doctors were less likely to do this than men.

It is worrying that the findings of this study and those of Dr Shapiro show some indifference to major issues regarding patients' rights such as informed consent and anonymous testing. This is particularly sad given the crucial role that general practitioners will have in the future in caring for those with AIDS or HIV infection and their families. Such indifference will inevitably lead to mistrust on the part of patients and to a reluctance to seek help from general practitioners when it is needed. Future efforts in educating general practitioners about HIV must address these matters.

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1 Shapiro JA. General practitioners' attitudes towards AIDS and their perceived information needs. *Br Med J* 1989;298:1563-6. (10 June.)

Passive smoking and middle ear effusion in children

SIR,—Dr D Strachan and colleagues presented an interesting report on the possible association of passive smoking with otitis media with effusion,[1] but there are three points that need addressing before their conclusions can be reached.

Eustachian tube dysfunction is extremely common in children and gives rise to negative middle ear pressure and middle ear effusion. The prevalence of middle ear effusion varies inversely with age, but there is also a marked seasonal variation, possibly related to a similar variation in upper respiratory tract infections.[2] The authors have quite rightly confined themselves to a single age group, but they carried out their tests over a period from January to June. Those tested earlier should have a higher rate of abnormal results, but this is not taken into account in the analysis.

Perhaps more importantly they fail to indicate whether the children had already had ear, nose, and throat operations (which at the age of 7 must be a considerable percentage) or whether there were other important factors such as cleft palate or Down's syndrome.

As otolaryngologists we deplore the use of tympanometry alone in the diagnosis of middle ear disease. It is a useful screening test, but in the presence of an abnormal finding we believe otoscopy must be performed. Frequently an obvious cause for the flat tympanogram such as wax, perforation, or even a grommet will be found. Dr Strachan and colleagues unfortunately do not seem to have checked their findings with otoscopy.

A possible indicator of this fault can be found in their numbers of abnormal tympanograms with increasing cotinine concentrations. There seems to be a trend relating increasing incidence of flat tympanograms to cotinine concentration, but there is no such trend with negative middle ear pressure. If one assumes that the same pathological process causes both negative middle ear pressure and middle ear effusion through dysfunction of the eustachian tube then the association between passive smoking and middle ear effusion is quite likely to be spurious.

It seems a pity that a paper written by an epidemiologist, a psychiatrist, and a chemist about an ear, nose, and throat condition should not have had the very necessary skills of an otolaryngologist to validate its findings.

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1 Strachan DP, Jarvis MJ, Feyerabend C. Passive smoking, salivary cotinine concentrations, and middle ear effusion in 7 year old children. *Br Med J* 1989;298:1549-52. (10 June.)
2 Hinchcliffe R, Prasansuk S. Epidemiology and SOM. A review. *Scand Audiol [Suppl]* 1986;26:53-8.

AUTHORS' REPLY,—Impedance tympanometry may not be the definitive diagnostic test for middle ear diffusion, but most previous epidemiological surveys have relied on this technique. Tympanometric measurement of the physical volume of the ear canal guards against the common sources of error. High values (>2·0 ml) indicate a perforated eardrum or patent ventilation tube, and in the seven children with such abnormality we conservatively chose to analyse the tympanogram from the other ear. No results were recorded from ears with blockage of the probe or low physical volume (<0·5 ml) suggesting wax. It is unlikely that flat tympanograms attributable to impacted wax could have generated a spurious association with passive exposure to smoke.

Children whose tonsils or adenoids had been removed (n=104) were at substantially higher risk of middle ear effusion (22% *v* 8%). Such a history was unrelated to the presence of smokers in the household (12% *v* 13%), so it is unlikely that previous surgical treatment affected the observed relation between middle ear effusion and passive exposure to smoke.

Different relations of passive smoking to type C and type B tympanograms might be expected if tobacco smoke affects the persistence of effusions rather than their incidence. In fact, normal (type A) tympanograms were less common in the children with higher cotinine concentrations, so that among the children without effusion there was a slightly higher risk of reduced middle ear pressure with heavy exposure to smoke (table II in our paper).

Month of examination would not affect the association between tympanometric findings and the number of smokers in the household, which was ascertained by a simultaneous questionnaire survey, but it was a potential confounding variable in our analysis of middle ear effusion and salivary cotinine concentrations. The prevalence of type B tympanograms was higher among children tested in January or February (12%) than in March or April (10%) and May or June (7%). After adjustment for sex, housing tenure, and number of smokers in the household the geometric mean salivary cotinine concentration in January and February was approximately double that in May and June. Nevertheless, the relation between middle ear effusion and the logarithm of the cotinine concentration remained significant after adjustment for month of examination (odds ratio per doubling 1·12, 95% confidence interval 1·01 to 1·25, $\chi^1=4·30$; df=1). Indeed, after adjustment for log cotinine the trend in prevalence by month of examination was non-significant (χ^1 (trend)= 1·07, df=1). Greater indoor exposure to tobacco smoke during the winter may contribute to the seasonal variation in prevalence of middle ear effusion.

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SIR,—I agree with Dr D P Strachan and colleagues that middle ear effusions in children should be recognised as one of the hazards of passive smoking.[1] It has been recognised for some time that passive smoking may have deleterious effects on the respiratory tracts of children. It is only recently that the adverse effects of passive smoking on the middle ear have been reported.

Not only is passive smoking in children associated with a higher than expected incidence of middle ear effusions and abnormal results of tympanometry[2] but these children are put at an increased risk of undergoing surgery for the condition. There is an increased incidence of grommet insertion and adenoidectomy in children whose parents smoke. Such children are twice as likely to require adenoidectomy, and their chance of requiring grommet insertion is increased by half.[3]

It is important to impress upon smoking parents that they may be subjecting their children not only to a greater risk of middle ear effusions but also to an increased likelihood of surgical intervention for the condition with the possibilities of both physical and psychological complications.

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Transurethral prostatic resection: a safe operation

SIR,—I was intrigued by Mr G Williams and colleagues' use of an expandable metal mesh stent for treating prostatic obstruction in patients considered unfit for transurethral prostatic surgery.[1] I was surprised, however, by their statement that the mortality associated with transurethral resection of the prostate has "led to a search for less invasive treatments," thus prompting this innovation.

Equally surprisingly, Mr Williams and colleagues were presented (in a short time, it would seem) with nine patients who were considered unfit for prostatic resection and hence were offered a stent as alternative treatment. Although the authors did not indicate the timespan over which these cases were collected, it could not have been very great as the first urological use of these stents was reported only in 1988. Accordingly the nine patients considered unfit for transurethral resection would seem to represent an uncharacteristically high proportion of all patients referred for prostatic surgery during this relatively short period. All this is at odds with my experience and that of my colleagues. I work in a 590 bed teaching hospital. In the 12 months to March 1989, 328 transurethral resections of the prostate were performed by the urology unit. None of the patients died, and only one patient with severe ischaemic heart disease was advised that he was unfit for surgery.

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